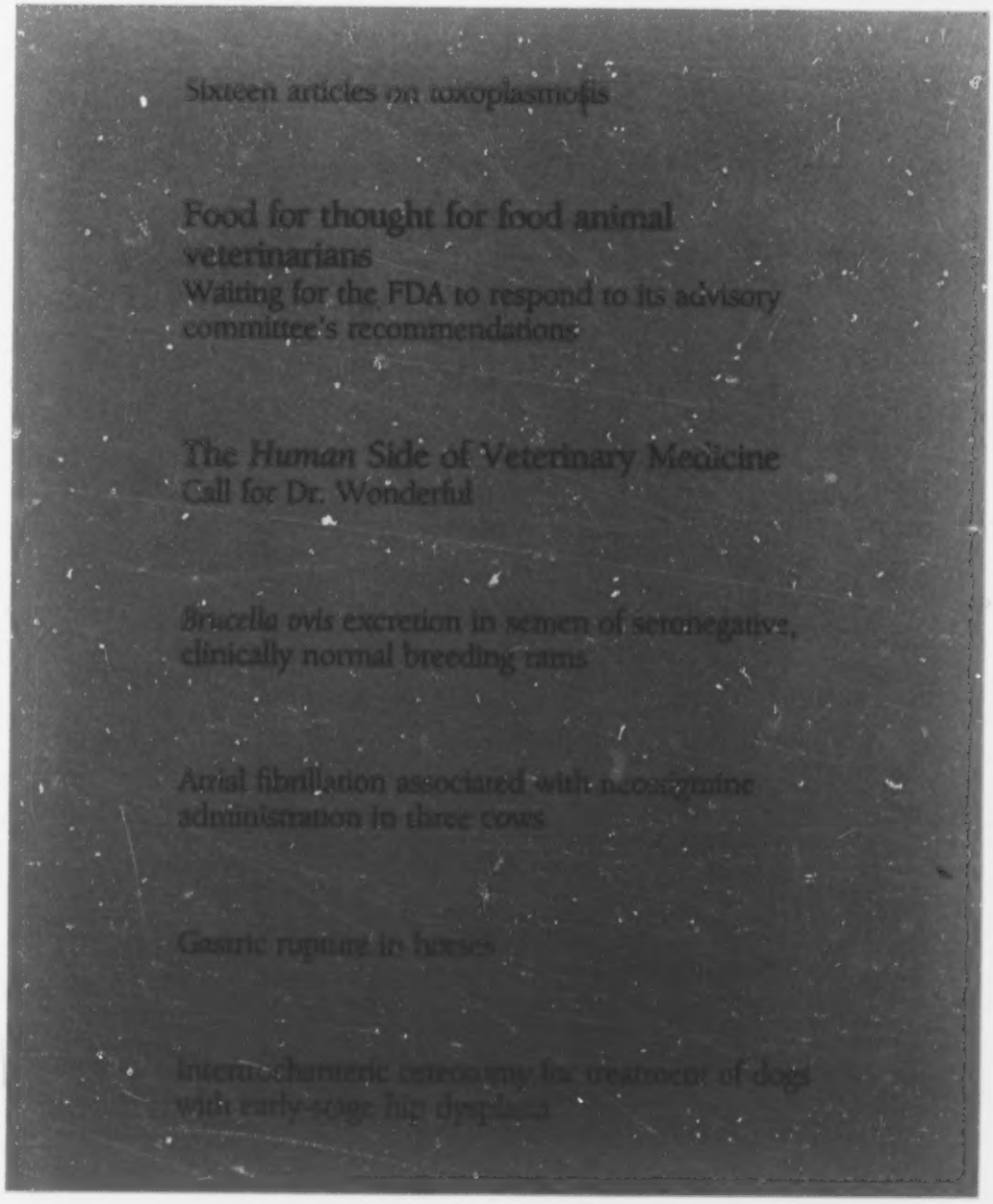
A3100-2

January 15, 1990, Volume 196, Number 2

JOURNAL

OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION



Sixteen articles on toxoplasmosis

Food for thought for food animal veterinarians
Waiting for the FDA to respond to its advisory committee's recommendations

The *Human* Side of Veterinary Medicine
Call for Dr. Wonderful

Brucella ovis excretion in semen of seronegative, clinically normal breeding rams

Atrial fibrillation associated with neostigmine administration in three cows

Gastric rupture in horses

Intramedullary centesis for treatment of dogs with early-stage hip dysplasia

JOURNAL
OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

© American Veterinary Medical Association, 1990
All Rights Reserved

Vol 196 January 15, 1990 No. 2
JAVMA 196 (2) 177-384 (1990)
ISSN 0003-1488

Interpretive Summaries of Articles in This Issue 180

Letters
More on Tufts' student surgery program 184
Chemical dependency 184
Clarification of a Legal Brief case 185

News
AVMA at Work—Board takes actions at fall meeting 187
Animal welfare concerns addressed 190
Cats should be vaccinated for rabies 191
AVMA offers new membership services 192
Single convention registration fee 193
Edge of Education—Interinstitutional Veterinary Cytology Resource Center developed at Purdue 195
Under the Umbrella—AABP members converge in Kansas City 197
Practice Management Corner—Staff meetings open doors to communication 200
Regulatory Front—Growth enhancers: boon or boomerang for the pork industry? 201
Technicians Today 203
Your Auxiliary in Action 204
Awards and Honors 206
Out and About 207
Deaths 208

Food for thought for food animal veterinarians
Waiting for the Food and Drug Administration to respond to its advisory committee's recommendations—*John B. Herrick* 222

AVMA Guide to Hazard Communication ..... insert

Difficult Dermatologic Diagnosis
Stephen D. White 225

Pages from the Past
Diseases of the horse. The centennial of a great book—*Franklin M. Loew* 226

The *Human* Side of Veterinary Medicine
Call for Dr. Wonderful—*Jacob Antelyes* 229

Reports from the Scientific Program, Section on Toxoplasmosis, 126th Annual AVMA Meeting, Orlando, Fla, July 15-19, 1989
Transmission of toxoplasmosis and the role of immunity in limiting transmission and illness—*J. K. Frenkel* 233
Toxoplasmosis in human beings
J. K. Frenkel 240
Estimating income losses and other preventable costs caused by congenital toxoplasmosis in people in the United States—*Tanya Roberts and J. K. Frenkel* 249
Status of toxoplasmosis in cattle in the United States—*J. P. Dubey* 257
Status of toxoplasmosis in sheep and goats in the United States—*J. P. Dubey* 259
Toxoplasmosis in sheep in northeastern United States—*Masoud A. Malik, David W. Dreesen, and Armah de la Cruz* 263
Prevalence of toxoplasmosis in swine from Iowa—*Jeff J. Zimmerman, David W. Dreesen, William J. Owen, and George W. Beran* 266
Status of toxoplasmosis in pigs in the United States—*J. P. Dubey* 270
Toxoplasma gondii infections in wildlife
David W. Dreesen 274
Serologic aspects of toxoplasmosis
Marianna Wilson, Doris A. Ware, and Dennis D. Juranek 277
Strategies for control of toxoplasmosis
J. C. Leighty 281

Reports of Original Studies
Toxoplasmosis and other causes of abortions in sheep from north central United States
J. P. Dubey and C. A. Kirkbride 287
Serologic and histologic diagnosis of toxoplasmic abortions in sheep in Oregon—*J. P. Dubey, R. J. Sonn, O. Hedstrom, S. P. Snyder, and E. D. Lassen* 291
Prevalence of *Toxoplasma gondii* antibodies in dairy goats from 1982 to 1984
J. P. Dubey and D. S. Adams 295
Diagnostic ultrasonography for evaluation of cryptorchidism in horses—*Henry W. Jann and Jerry R. Rains* 297
Seroprevalence of *Actinobacillus (Haemophilus)* pleuropneumoniae serotype-1 infection in swine herds in Quebec—*Jean-Pierre Vaillancourt, Guy-Pierre Martineau, Serge Larivière, Robert Higgins, and Khyali R. Mittal* 301

Evaluation of an automated tabletop blood biochemical analyzer for the veterinary clinical pathology laboratory—*Susan Sallee, Ray Rippel, and Walter E. Hoffmann* 307

Brucella ovis excretion in semen of seronegative, clinically normal breeding rams—*Marie S. Bulgin* . 313

Clinical Reports

Myelitis in a cat infected with *Toxoplasma gondii* and feline immunodeficiency virus—*J. R. Heidel, J. P. Dubey, L. L. Blythe, L. L. Walker, J. R. Duimstra, and J. S. Jordan* 316

Total parenteral nutrition in a neonatal llama *L. R. Hovda, S. M. McGuirk, and D. P. Lunn* 319

Systemic amyloidosis in a mare *Tracy Bennett Hawthorne, Brad Bolon, D. J. Meyer* . 323

Intestinal adenocarcinoma in a goat *George K. Haibel* 326

Atrial fibrillation associated with neostigmine administration in three cows—*Peter D. Constable, William W. Muir III, Lisa Freeman, Glen F. Hoffsis, Guy St. Jean, and Frank H. Welker* . 329

Reports of Retrospective Studies

Gastric rupture in horses: 50 cases (1979-1987) *Mary Lou Kiper, Josie Traub-Dargatz, and Charles R. Curtis* 333

Clinical evaluation of intertrochanteric osteotomy for treatment of dogs with early-stage hip dysplasia: 37 cases (1980-1987) *T. D. Braden, W. D. Prieur, and J. B. Kanecne* 337

Reports on Wildlife and Laboratory Animals

Comparison of serum and plasma taurine values in Bengal tigers with values in taurine-sufficient and -deficient domestic cats—*J. Phillip Pickett, Russell W. Chesney, Bruce Beehler, Cecil P. Moore, Shirley Lippincott, John Sturman, Kerry L. Ketring* . 342

Acute toxoplasmosis in a camel—*W. A. Hagemoser, J. P. Dubey, and J. R. Thompson* 347

Economic Note

Trend in real starting salaries of US veterinary medical college graduates, 1980-1989 *J. Karl Wise* 348

What Is Your Diagnosis?

Roberta Wallace 351

Book Reviews

Problems in Equine Medicine 300
Diagnostic Cytology of the Dog and Cat 349
Medicine and Surgery of South American Camelids: Llama, Alpaca,Vicuna, Guanaco 352
Books received but not reviewed 328

Announcements

Coming Meetings 354
Foreign Meetings 356
Applications for Membership 356
Certification Examinations 358
Educational Opportunities 359

Classified Ads 363

Coming Soon . . .

February 1

Reflections—The path followed . . . (J. J. Mettler, Jr.)

Legal Brief—Advertising and ethics—is there still a relation? (H. W. Hannah)

Carbon dioxide laser ablation for treatment of limbal squamous cell carcinoma in horses (R. V. English)

Evaluation of upper respiratory tract function during strenuous exercise in racehorses (E. A. Morris and H. J. Seeherman)

February 15

Food for thought for food animal veterinarians—It works in my practice (J. B. Herrick)

Special Commentary—Abbreviations and neologisms in clinical veterinary medical education: Does everyone understand what we are saying? (B. S. Jortner et al)

The *Human* Side of Veterinary Medicine—Client relations when the animal dominates (J. Antelyes)

Protozoal encephalomyelitis in horses (M. G. Boy et al)

Antibody production in swine vaccinated with *Actinobacillus* bacterins containing various adjuvants (B. E. Straw et al)

Randomized blind trial of a commercial FeLV vaccine (R. V. H. Pollock and J. M. Scarlett)

Estimating income losses and other preventable costs caused by congenital toxoplasmosis in people in the United States

Tanya Roberts, PhD, and J. K. Frenkel, MD, PhD

Previous estimates of costs of congenital toxoplasmosis in the United States have focused on medical expenses and special education or residential care for visually impaired and mentally retarded survivors.[1,2] In the United States, costs have not been estimated for income losses for fetuses or infants who become mentally retarded and have reduced lifetime income or who die as a result of congenital toxoplasmosis. In the United Kingdom, research workers have included these income losses in their estimates of the cost of toxoplasmosis.[3]

Estimated number of annual cases

Reported annual estimates of congenital toxoplasmosis in the United States[4-7] differ because studies have examined different parts of the US population and exposure to *Toxoplasma gondii* may vary. Differences in exposure may take place because of cultural food preparation habits that encourage contact with raw pork or consumption of rare pork. Most cases are thought to result from handling raw meat or eating undercooked pork and, to a lesser extent, mutton, venison, beef, and chicken. There also are differences in climate affecting organism survival or differences in contact with cats or soil contaminated with cat feces.

The probability of maternal and fetal infection depends on the time at which infection develops in the pregnancy.[8,9] Even if exposure and maternal and fetal infection rates were identical, research workers may find different infection rates because they use serotests that differ in their sensitivity and specificity to determine the presence of *T gondii* infection.[10,11] Another factor may be changes in exposure over time because of the increasing use of preventive actions (washing hands after contact with raw meat, garden soil, or cat litter and before eating) by pregnant women to reduce exposure, food manufacturer's increased freezing of meat, which kills most cysts; increasing use of swine confinement pens that may limit cat access and reduce disease transmission to swine; or changing patterns of cat ownership and cats' diet.[12]

Table 1—Calculation of regional and national Toxoplasma *exposure in human beings*

Region	Regional exposure (%)	Share of population (%)	Contribution to US exposure (%)
Northeast	20	5.27	1.05
Mid Atlantic	16	15.48	2.48
East north central	18	17.30	3.11
West north central	12	7.30	0.88
South Atlantic	18	16.97	3.05
East south central	19	6.31	1.20
West south central	13	11.16	1.45
Mountain	3	5.39	0.16
Pacific	8	14.81	1.19
Total	...	100	14.60

Exposure data based on 17- to 23-year-old recruits[5] and population data.[15]

Because of the uncertainty associated with estimating the number of maternal and fetal infections, 2 methods are used to develop estimates. One approach examines possible maternal exposure and the likelihood of transmission to the fetus to estimate congenital exposure. Nationwide exposure data are sketchy. Data from Sever et al[13] are not used because of the lack of systematic follow-up examination of the babies to verify how many were really infected with *Toxoplasma*. The most extensive data on geographic variation in exposure come from a survey of military recruits.[5] Feldman[5] estimated the cumulative exposure for 17- to 23-year-old recruits in various states and combined data into regional exposure rates that range from 3 to 20%. Weighing these rates by current regional population yielded a national average exposure of 14.6% (Table 1). Assuming pregnant women have similar exposure patterns, the national average cumulative exposure for pregnant women would be similar (14 to 15%).

Using cumulative exposure data, Frenkel[1] estimated annual seroconversion rates indicating annual exposure. The 14 to 15% cumulative exposure rate for 17- to 23-year-olds translates to an annual seroconversion rate of 0.5 to 1%, depend-

From the Economic Research Service, USDA, 1301 New York Ave, Washington, DC 20005-4788 (Roberts) and the Department of Pathology and Oncology, College of Medicine, University of Kansas Medical Center, Kansas City, KS 66103 (Frenkel).

The authors thank David Smallwood and George Frivsold, USDA Economic Research Service, and Drs. Rima McLeod, Michael Reese Medical Center, University of Chicago; Ph. Thulliez, Institute de Puericulture de Paris; Rodney Hoff, Massachusetts Laboratory Institute; C. B. Wilson, University of Washington, and Larry Mars, Rehabilitation Service Administration, US Department of Education, Washington, DC, for technical assistance.

Table 2—Estimated number of annual fetal infections as a function of maternal infection and the maternal/fetal transmission rate in human beings

Maternal/fetal transmission rate (%)	Maternal seroconversion rate	
	0.5%	1%
30	4,200	8,400
45	6,300	12,600
60	8,400	16,800

Mean (rounded to nearest 100) is 9,500 cases/yr.

ing on how one adjusts for the relative lack of exposure before the ages of 5 to 10 in the United States. Contrary to data from Central America, where *Toxoplasma* infection is actively acquired during early childhood,[14] transmission in the United States starts later.[15] Therefore, the prevalence rates for 20-year-olds cannot be interpolated to 0% at birth, but are more likely to be 0% between ages 5 and 10. The total number of maternal exposures each year is then estimated by multiplying the annual 3.7 million live births[16] times the annual maternal seroconversion rate times 9/12 (portion of the year a woman is pregnant). With a seroconversion rate of 0.5%, total estimated maternal exposures are 14,000/yr. With a seroconversion rate of 1%, twice as many maternal exposures take place (28,000/yr).

However, not all maternal exposures are transmitted to the fetus. The range varies from a fetal exposure 33% of the time[9] to 61%.[8] In one study,[9] some mothers were treated with spiramycin, reducing the rate of transmission to the fetus. However, both estimates reveal how the estimated number of annual congenital infections varies with the maternal infection rate and the maternal/fetal transmission rate (Table 2). Estimates of fetal infections range from 4,200 to 16,800 cases/yr. Assuming each estimate has an equal probability of occurring, a reasonable population estimate is the simple average of all 4 estimates, or 9,500 congenital infections.

Another approach to estimating the number of infections is based on serotests of blood samples from newborns. In Massachusetts,[a] capillary blood was collected from newborns (usually 3 days old) and examined for IgM *Toxoplasma* antibodies. The Massachusetts data could represent an area of the United States with a relatively high incidence rate. However, the maternal prevalence in a substudy of the Massachusetts study was 14%, which is quite similar to the US incidence (Table 1). Preliminary findings indicated *Toxoplasma* antibodies, and therefore infection, in only 1 in 10,000 babies. This estimate is increased by 10% to account for the possibility that the IgM test used was not as sensitive as the IgA test. Multiplying this estimate by the live-birth rate in the United States of 3.7 million/yr[16] results in an estimated 407 cases of congenital toxoplasmosis/yr.

[a]Hoff R, State Laboratory Institute, Massachusetts Department of Health, Boston, Mass: Personal communication, 1989.

However, only paired testing for IgA and IgM can verify these assumed differences in test sensitivity. The sensitivity of the IgM test Hoff[a] used was greater than the IgM test used by Decoster et al,[10] although the difference has not been determined concretely. For the 26 infants whose mothers were infected during pregnancy,[10] IgA antibodies were detected in 8, although only 3 infants also had IgM antibodies. This fact may indicate that the IgA test is more sensitive by a ratio of 8:3 (2.67) and may indicate that the Hoff findings[a] could be doubled. However, the Decoster et al[10] findings are complicated by inclusion of some older babies in whom IgM titers may have decayed.

In the 1960s, 1 of 486 babies had increased IgM values in serum obtained from umbilical cord, indicating *Toxoplasma* exposure.[17] In the next year, the incidence was 1 of 1,500, for a mean incidence over the 2 years of 1 of 1,000. In the 1980s, the incidence declined to 1 of 10,000.[b] Although these 2 methods of computing the likely toxoplasmosis infections produce different results (407 vs 9,500 estimated infections), the range cannot be narrowed until national screening serotests are conducted.

Effects of congenital toxoplasmosis

Consequences of *T gondii* infection in fetuses and newborns can only be estimated by prospective studies in which asymptomatic infants and infants with symptoms of illness are monitored. Otherwise, mild or subclinical cases will be missed and only severe cases will be estimated. The largest prospective study was conducted in France, where newborns are routinely tested for *Toxoplasma* antibodies indicating exposure.[18] To adapt the French study to the United States, only data for nontreated maternal infections were used. In the United States, most maternal infections are asymptomatic or undiagnosed.[2,19] In France and Austria, testing for *Toxoplasma* during pregnancy is mandatory, but the low incidence of infection in the United States results in only sporadic testing of a small fraction of pregnant women. Few adults with toxoplasmosis seek treatment for the mild symptoms. In one study,[20] only one person sought treatment for a high fever and headache, although in a subsequent epidemiologic investigation, 36 others had high titers indicating *Toxoplasma* exposure. Therefore, only 1 of 37 persons sought medical attention for symptoms of infection. A collaborative study in the United States to determine consequences of infection and optimal treatment is just being started.

For fetal exposure, 2% of infants die[18]; this is a doubling of deaths to account for the underreporting in that study, because infants who had already died were not part of the study (Table 3).

[b]Alford CA, Department of Pediatrics and Clinical Pathology, University of Alabama Medical Center, Birmingham, Ala: Personal communication, 1989.

Spontaneous abortions[21] were left out of estimated cases because adequate data were not available.

Most studies[22,23] are based on clinically selected cases that emphasize the severe and overlook the mild cases that are better represented in prospective studies. Later sequelae of congenital toxoplasmosis have been studied prospectively[24] in children until age 11 to 21 years. The most common consequences were severe mental retardation in 20% of cases, moderate visual impairment, 20%; strabismus or cross-eyed vision, 13%; slight mental retardation, 12%; moderate hearing loss in one ear, 10%; and moderate mental retardation, 9% (Table 3).

Table 3—Impairments caused by congenital toxoplasmosis in human beings

Impairment	Percentage			
	First year[18]	Additional in second year[18]	3 to 17 years[24]†	Total affected (%)
Death	2	0	0	2
Severe illness	11	0	0	11
Severe retardation	11	+2	+20	33
Moderate retardation	5	+3	+9	17
Slight retardation	5	+6	+12	23
Blindness (bilateral)	6	0	+2	8
Moderate visual impairment	16	+17	+20	53
Strabismus	5	+2	+13	20
Deafness	2	0	0	2
Hearing loss (moderate unilateral)	0	0	+10	10
Normal	55*	36*	20*	20*

*After 20 years. †A few lesions were present at the time of the first visit. Some original percentages were increased based on clinical and epidemiologic suspicions, given the rarity of making a diagnosis in the United States outside of specialty medical centers: severe retardation was increased from 8 to 20%, moderate retardation was increased from 8 to 9%, and bilateral and unilateral blindnesses were combined for a 2% estimate.

Deafness varied widely from 0 to 13%.

Estimated economic costs

Three types of costs were estimated: income losses because of impairment or death, reducing the potential labor market participation and/or income of affected individuals; medical costs incurred from infancy through adulthood; and special education costs or residential care required because of a handicap caused by congenital toxoplasmosis.

Lost income—Income losses because of impairment or death are unemployment, working in a lower-skill job for lower pay than an unimpaired individual, or death.

Valuing the loss of an infant's life to society is a subjective issue. Is the loss of a fetus valued less than that of an infant who lives for a short time? If the fetus or infant dies, how often will it be replaced by another pregnancy? Parents could be considered to value a child at least as much as they would pay to raise the child. Or we could consider that if the fetus or infant had lived, an average work history would have occurred and the income loss is equal to a lifetime of production or income. This option of valuing lifetime income, called the human capital method, is the economists' traditional method for valuing a shortened life. The lifetime stream of income is converted to a present-day value by discounting future income to its worth today, given today's real rate of interest. The most recently suggested method[25] estimates what people will pay to reduce risks to life; these willingness to pay estimates are the highest of all.

The present value of income losses were estimated, using a modified traditional human capital equation,[26] which defines income more broadly and adds a risk aversion factor:

$$\text{Present value of reducing risks to life} = \left[\sum_{t=0}^{T} \frac{Y_t}{(1+r)^t}\right]\alpha$$

Income, Y, is after-tax labor and nonlabor income (measured in constant dollars so inflation is taken out of the estimates) as well as home-produced good and services, such as meals. T is time or the person's remaining lifetime. A specific year is t. The real rate of interest, r, used to discount future income to today's value, is 3%/yr. Again, the interest rate does not include inflation. A 3% discount rate represents a household's real rate of return on investment. Consensus is that the US government's opportunity cost of capital is the international real rate of interest. Rates of 2 to 4% have been discussed and explicitly mentioned as rates that should be used in benefit/cost analysis.[c]

The addition of the risk aversion factor, α, is based on data on life insurance purchases that reveal people with life insurance pay 60% extra in premiums over the probable lifetime income loss. This extra payment represents the relationship between the willingness to pay for reduced risk (the outlay for insurance) and the expected benefits of the insurance (the probability of a loss multiplied by the value of the asset in question). The extra 60% reflects the risk aversion benefit of protecting a household income. All t variables in the equation are derived from market valuation of goods and services produced or purchased during one's lifetime. Updated to 1989 dollars,[27] (using the increase in the average weekly earnings for production workers reported by the Bureau of Labor Statistics) the estimated present value of an infant's life is $983,000.

Norwegian data[28] have been evaluated for the likelihood that a stillbirth or infant death will be replaced by another pregnancy. Although the likelihood varies by maternal age, on average, a replacement child is conceived 60% of the time. We assume that families in the United States respond similarly. Thus, for fetal or infant deaths, we assume that 60% of the deaths will be replaced by siblings who fully compensate for the earlier loss and that 40% will not be replaced. In other words,

[c]Allied Social Sciences Association, New York, NY: Unpublished data, 1989.

Table 4—Effect of retardation on employment and loss of income[31]

Degree of retardation	Men (%)				Women (%)			
	Working	Income compared with US average	Not working	Total loss*	Working	Income compared with US average	Not working	Total loss*
Slight (IQ 50 to 69)	87	86	4	18	33	87	12	36
Moderate (IQ 40 to 49)	45	19	46	91	12	19	33	95
Severe (IQ <40)	0	0	100	100	0	0	100	100

*The total loss in income of the retarded group is the loss of the income for those retardates not able to work plus the reduced income of working retardates. The equation: Total percentage of loss = [(percentage of retardates not working/percentage working in general population) × 100%] + [(percentage of retardates working/percentage working in general population) × (1-percentage of income)].

In this data set, 91% of men and 45% of the women in the general population were working.

for the fetal or infant death category, cases are valued at 40% of the income loss. Emotional cost of losing a fetus or baby,[29,30] the maternal cost of being hospitalized for a stillbirth or subsequent infant death, and the cost of a delay in having a replacement child are all omitted from the estimates. In some cases, these costs may be important.

In an exhaustive study[31] of costs of mental retardation in the United States in 1973, slightly retarded adults (IQ, 50 to 70) were able to find jobs at wages close to the average income of the general population (Table 4). Moderately retarded persons (IQ, 40 to 49) frequently found jobs, but their wages were only 19% of the average. Few retardates with IQs below 40 were employed. Combining the income loss as a result of lower wages and the loss as a result of greater unemployment, we have calculated the total income loss for slightly retarded men at 18%, for slightly retarded women at 36%, for moderately retarded men at 91%, for moderately retarded women at 95%, and for the severely retarded of both genders at 100%.

Effects of mental retardation on income have been estimated[3] (Table 5). Data assumed the severely retarded would not be employable and would have a 100% income loss. For the moderately retarded group, a 67% income loss, was assumed,[3] whereas another analysis[31] revealed > 90% income loss. For the slightly retarded, women's reduced incomes were comparable in both estimates, but for men, Conley[31] estimated only an 18% income loss, which is less than the 33% reported elsewhere.[3]

Other data are available for those mentally retarded individuals, exiting state/federal programs of vocational rehabilitation.[d] In 1983, 26,000 retardates successfully completed rehabilitation services. Data for those entering the program revealed 92 to 93% of retardates at all 3 degrees of retardation were unemployed and most were under 25 years old. On completion of the program, 93 to 94% (at all 3 degrees of retardation) entered the paid labor force, although placement in sheltered workshops increased dramatically as the degree of retardation increased. The income profile is flatter for this data set than for the others: for the slightly retarded, income loss was 49% of the median weekly income of the general population; for the moderately retarded, income loss was 63%; and the severely retarded had a 79% earning loss.

Degrees of retardation used in the Department of Education's retraining program are a combination of IQ and behavior and are not strictly comparable with categories[31] based only on IQ. Earnings of people exiting training are compared with earnings of the younger members of the labor force, which are 67% of the median weekly earnings.[16]

[d]Mars L, Rehabilitation Services Administration, US Department of Education, Washington, DC: Unpublished data, 1989.

Table 5—Comparison of studies on estimated income reduction (%) because of mental retardation

Retardation	Group A[3]	Group B[31]		Group C[a]	Group D[a]	Group E[42,*]
		Men	Women			
Slight	33	18	36	27	49	10 to 50
Moderate	67	91	95	93	63	
Severe	100	100	100	100	79	

*For small deviations around the mean IQ (ie, 100), the authors concluded that a 1% decrease in IQ has been associated with around a 1% decrease in income, with the range between 0.24% and 1.25%.[42] For a reduction in IQ of 40 points (slight retardation), this translates to a decrease in income of 10 to 50% that brackets other estimates of income loss. The 40-point change is relatively large and may be stretching the analysis to extrapolate the income change this far.

Group-C data are an adaptation of the group-B data, in which babies with congenital toxoplasmosis are assumed to be equally allocated between genders.

Table 6—Income losses for deaths and impaired survivors (low estimate assuming 407 congenital cases annually and high estimate assuming 9,500 cases)

Outcomes	Income loss/case ($)*	No. of cases		Total (million $)	
		Low	High	Low	High
Fetal/perinatal deaths†	393,200	8	190	3.1	74.7
Severely retarded	983,000	134	3,135	131.7	3,081.7
Moderately retarded	914,200	69	1,615	63.1	1,476.4
Slightly retarded	265,400	94	2,185	24.9	579.9
Blind	688,100	33	760	22.7	523.0
Deaf	550,500	8	190	4.4	104.6
Total loss				249.9	5,840.3

*The income loss for deaths is 40%, because 40% of infants are not replaced by another birth. The income loss is 100% for the severely retarded because they do not work. The income loss for the moderately retarded is 93%, the slightly retarded 27%, the blind 70%, and the deaf 56%, reflecting their average income compared to the average US population. †Newborn deaths, stillbirths, and fetal losses are assumed to be replaced by another child 60% of the time. If only 50% are replaced, the estimated fetal/perinatal loss would be increased to $4.1 to $99.6 million. If 70% are replaced, the estimate would be decreased to $2.5 to $59.8 million.

Income losses are not adjusted for double counting because cases falling into 2 categories are likely to be penalized for both disabilities.

Table 7—Special education and residential care costs for mentally retarded people

Congenital toxoplasmosis outcomes	Present value of costs/case ($)*	No. of cases		Total costs (million $)	
		Low	High	Low	High
Severely retarded (40-year life-span)					
Residential care	1,030,000	67	1,568	69.0	1,615.0
Live at home for school years†	109,000	67	1,568	7.3	170.9
and in institution	10,900			0.7	17.1
after age 18	417,142			27.9	654.1
Moderately retarded	77,000	69	1,615	5.3	124.4
Slightly retarded	43,000	94	2,185	4.0	94.0
Blind					
Special education	161,000	33	760	5.3	122.4
Additional family expense	161,000			0.5	12.2
Deaf					
Special education	113,500	8	190	0.9	21.6
Additional family expense	11,350			0.1	2.2
Total		338	7,886	115.7	2,833.9

*All costs are discounted by a 3% annual interest rate to arrive at a present value. †The top figure is the cost of schooling for the first 18 years at home, the second number is 10% extra for the family's additional expenses, and the third number is the cost of residential care after age 18.

All blind and deaf children are assumed to live at home instead of the more expensive residential care. This conservative assumption is made to offset any double counting that may occur because of multiple handicaps.

Average earnings for the retrained in 1983 were $108.10 for the mildly affected group, $78.7 for the moderately affected group, and $44.40 for the severely affected group.[d]

Self-selection of people entering training programs may explain much of the difference from Conley's data:[31] slightly retarded persons may be able to find jobs on their own, those who are less capable enter federal job training, and the most severely retarded may not apply for or be selected for federal job training. The youthfullness of the groups may also be important. Still, the range of income losses is close to other estimates.[3,31] Because Conley's data[31] are the most comprehensive, his data on reduced income were used for the estimated income losses caused by mental retardation (Table 6).

For income losses caused by other impairments, a study at Rutgers was used.[32] The income loss of persons with visual impairment was 70%, and the hearing impaired had a 56% income loss (Table 6). Because of the small number of people with these impairments, adding these income losses did not increase the estimates. Most income loss is a result of severe or moderate mental retardation. The large range in the estimates, from $0.2 to 5.8 billion, is a result of different estimates of the number of cases of congenital toxoplasmosis each year.

Costs of special education and residential care—Half of the severely retarded are assumed to be living in institutions and the other half to be living at home.[3] The annual average expenditure for public education was $3,970[16]/student in public school in 1987. Because 88% of children in the United States are in public schools, $3,970 was used as the baseline cost. One study[33] calculated the additional cost of educating persons with various disabilities. For educable mental retardates, the additional cost was 1.30 times the cost of regular education, (1.30 × $3,970 = $5,160/yr). For trainable retardates, the additional cost was 2.34 times the cost of regular education (2.34 × $3,970 = $9,290/yr). For the severely retarded, the additional cost was 2.59 times the cost of regular education (2.59 × $3,970 = $10,280/yr). Assume 12 years of education plus an additional 3 years of preschool for the severely retarded.[2] For residential cases, costs were assumed to be the average fee of $122/day for a mentally retarded person in a residential facility.[16] For the severely retarded living at home, and extra 10% was added to the education costs for additional home expenses.[3] All costs are discounted by a 3% real rate of interest to arrive at a present-day value.

Additional education costs for children who are deaf or blind are greater than for the severely mentally retarded: 3.43 and 4.86 times the average cost, respectively, for estimates of $13,617 and $19,294/yr, respectively. A 10% additional fee is added for a family's extra expenses, and the costs are discounted. The severely retarded dominate the estimates, largely because of the high cost of residential care. The range of total costs is $116 million to $2.8 billion (Table 7).

Medical costs—Medical costs are incurred by those ill as infants and those with visual impairment. For the 2% of infants who die, we assumed 3 weeks of intensive-care hospitalization at $1,000/d[34] and an equal amount for physician services, tests, and drug treatment,[35] for a cost of $42,000/case (Table 8). For the 11% of infants who survive severe illness, we assumed 2 weeks in intensive care at $1,000/d and 2 weeks in a regular hospital room at $500/d,[34] plus a fee equal to hospital costs for physician services, tests, and drug treatment[35] resulting in a cost of $42,000/case.

Because most children with *Toxoplasma* antibodies develop sequelae (Table 2), diagnostic tests to determine the cause of the clinical signs are re-

Table 8—Medical costs for congenital toxoplasmosis cases in human beings[c]

Outcomes	Cost/ case ($)	No. of cases		Total costs (million $)	
		Low	High	Low	High
Severe neonatal illness					
Deaths	42,000	8	190	0.3	8.0
Survivors	42,000	45	1,045	1.9	43.9
Diagnostic tests*	2,500	407	9,500	1.0	23.8
Yearly eye examination†	1,041	216	5,035	0.2	5.2
Strabismus‡	400	81	1,900		0.8
Total				2.4	57.9

*Each diagnostic work-up could cost around $2,000 to 3,000 (not discounted because most tests follow symptom development in first or second year of life). †Discounted at 3% annual interest to compute a present value. ‡Not discounted because of uncertainty over what year the operation would occur.

No estimate is made for the testing and physician time to diagnose congenital toxoplasmosis.

Table 9—Summary of preventable costs for congenital toxoplasmosis in human beings.

Cost category	Estimate (million $)	
	Low	High
Income loss	250	5,840
Special education/residential care	116	2,834
Medical costs	3	82
Total	369	8,756

Costs are discounted at 3%/yr.

quired. Generally these tests follow symptom development and are done in the first year or 2 of life. The estimated cost is $2,000 to $3,000/child.[e]

Those with visual impairment (48%) were assumed to have an annual eye examination for 20 years at cost of $70/examination.[f] Of these individuals, 17% were assumed to need an operation for strabismus (cross-eyed vision) at a cost of $400.[c] These medical costs for visual sequelae are small compared with the illness costs of hospitalized infants. The total medical costs are estimated to be $2.5 to 59 million (Table 8).

Of the 3 types of preventable costs estimated, income loss is two-thirds of the total cost (Table 9). The largest contributors to income loss are people who are severely or moderately retarded. Next in importance are special education and residential care costs. Medical costs comprise a small part of the estimated preventable losses. The range of total preventable cost estimates is from $368 million to $8.7 billion for the infants estimated to be born each year with congenital toxoplasmosis.

We believe the wide range in the estimates is caused by the great uncertainty over the actual number of cases. For social and economic reasons the incidence of toxoplasmosis in the United States may be declining and the order of magnitude of the lower estimate may be more accurate.

Finally, estimates do not include costs of toxoplasmosis to adults. Recrudescent toxoplasmosis has appeared in patients treated because of neoplasms and organ transplants, and in patients with acquired immune deficiency syndrome.

[e]Cho C, University of Kansas Medical Center, Kansas City, Kan: Personal communication, 1989.

[f]Godfrey W, University of Kansas Medical Center, Kansas City, Kan: Personal communication, 1989.

Discussion

Previous estimates have used different procedures or estimated different costs (Table 10). Lifetime costs[2] of special services for 3,300 children with congenital toxoplasmosis in the United States were estimated at $222 million in 1978. These figures did not include deaths, estimate income losses, or discount costs to compute a present value (which overstated costs), but did include a transfer payment of aid to the totally disabled that most economists would not include.

In the United Kingdom,[3] the greatest loss was a result of income loss and residential care for the moderately mentally retarded. Income loss is more narrowly defined than in the Landefeld and Seskin[26] measure used in this study. Costs are given in 1980 English pounds and are discounted at 5% to compute a present value. Over time, as cases of congenital toxoplasmosis in the United States are identified more quickly and treatment is started sooner, the incidence of sequelae can be expected to decline.

None of the studies,[2,3,31] including the present study, estimated psychological costs because of family disruption and the stress of having a mentally retarded child; nor did they place a value on the fact that the mentally retarded have fewer friends and are less likely to marry than are persons with an average IQ[31,36,37] (Table 10).

The wide range in the Roberts and Frenkel estimates reported here is a result of the great uncertainty over the number of infected babies. The difference in the estimated cases using information from mothers vs newborns may be partly a result of comparing data from people a generation apart. If the incidence of toxoplasmosis is declining, much of the accumulated incidence in the mothers reflects outdated socioeconomic circumstances, whereas data from recent newborns reflects recent socioeconomic factors (freezing of meat, changes in swine rearing, changes in cat ownership and diet, and greater handwashing after contact with raw meat) that cause a lower exposure rate.

Although routine testing of newborns would give us more data on today's actual incidence, establishing such a system nationwide should not be done without careful consideration of costs and the low incidence of infection. The predictive value of tests is a function of the incidence as well as the test sensitivity and specificity. We are accustomed to using the predictive values of tests in a clinical setting when cases are concentrated to between 1:10 (1 case/10 patients) and 1:100. However, when dealing with population surveys and a lower incidence of 1:1,000 or 1:10,000, as discussed here, the predictive value of even a sensitive and specific test drops to approximately zero.[38]

Table 10—Comparison of toxoplasmosis cost studies

Item	United States[7]	United Kingdom[3]	United States
Cases			
No. of congenital cases	3,300	195 to 585 to 1,950	407 to 9,500
Cases/1,000 live births	1.1/1,000	0.3/1,000 to 0.9/1,000 to 3/1,000	0.11/1,000 to 2.6/1,000
Assumed maternal/fetal transmission (%)	45	39	30 to 60; average, 45
Cost estimates			
Year	1978	1980	1987 to 1989
Annual discount rate	None used	5%	3%
Deaths (infant/fetal)	Nil	8% of cases	2% of cases
Medical costs	Nil	4 days in hospital	3 weeks of intensive care
Income loss	Nil	Nil	40% of lifetime income*
Severe illness at birth	8% of cases	10% of cases	11% of cases
Medical costs	Nil	4 weeks in hospital	2 weeks intensive care and 2 weeks regular hospitalization
Mental retardation			
Severely retarded	15.1% of cases	5% of cases	33% of cases
Residential care	40 years	½ for 10 years	½ for 40 years
Special education		½ for 6 years	½ for 15 years/live at home
Extra family expenses	None	£15/week†	10% of special education cost
Income loss	Nil	Total loss‖	Total loss‖
Moderately retarded	7.1% of cases	5% of cases	17% of cases
Special education	15 years	Primary and secondary	12 years
Extra family expenses	Nil	£15/week†	Nil
Income loss	2.4% unable to work (valued at disability payment for 44 weeks)	½ of lifetime income	93% of lifetime income
Slightly retarded	None	5% of cases	23% of cases
Special education		Primary and secondary	12 years
Income loss		½ of lifetime income	27% of lifetime income
Deafness	Nil	Nil	2% of cases
Special education	Nil	Nil	12 years
Extra family expenses	Nil	Nil	10% of special education costs
Income loss	Nil	Nil	56% of lifetime income
Visual Problems			
Special education for visually handicapped	14.2% for 10 years		Nil
Blind	¶	1% (adjusted to avoid double-counting with moderately retarded)	8% of cases
Special education		‡	12 years
Extra family expenses		Nil	10% of special education costs
Income loss		50% of lifetime income	70% of lifetime income
Yearly ophthalmologic follow-up care	78% for 20 years	20% for 6 years	53% for 20 years @ $70/examination
Operation for strabismus	Nil	46%	20% of cases @ $400

*Psychic loss to family is not estimated. †November 1980 price values. ‡Assumed ½ would be in residential care from 0 to 14 years of age and the rest would have extra costs for special education. §Included in severely retarded category. ‖Of lifetime income.
Nil = none estimated.

For benefit/cost studies, the relative cost of several proposals should be kept in mind. Four principal methods of preventing toxoplasmosis have been described: (1) repeated serologic screening during pregnancy, when necessary aided by amniocentesis, examination of fetal blood (culture and serologic) and ultrasonic examination, with chemotherapy/chemoprophylaxis and therapeutic/prophylactic abortion of fetuses believed or proven to be infected[2,39]; (2) serologic screening of newborns at the time of delivery,[40] together with testing for phenylketonuria and congenital hypothroidism, with treatment of newborns found to be infected, as practiced by Hoff[a]; (3) prevention of maternal infection by education on hygiene, stressing hand washing and adequate cooking of meat; and (4) controlling transmission of toxoplasmosis by cats, by vaccination combined with control of stray cats and of some intermediate hosts,[41] and in some circumstances vaccination of women of childbearing age who are likely to be exposed to *Toxoplasma*. The 4 strategies are compatible with one another, and all will probably come into use gradually over time. Still, if the expenditure of public funds were proposed in support of any of these approaches, cost-efficiency calculations would be desirable.

Although exact estimates depend on details of implementation of these proposed strategies, a tentative ranking of costs for the programs can be made on the basis of their relative complexity and labor intensiveness. In these terms, the cost would probably be stratified as 1 > 2 > 3 (multiple serologic, microbiologic, ultrasound examination during pregnancy > single serologic examination at birth > maternal education). The cost of the control of toxoplasmosis by vaccination of cats, and perhaps human beings, and other measures is still unknown and depends on the outcome of laboratory and field trials and on the cost of vaccine production and distribution.

References

1. Frenkel JK. *Toxoplasma* in and around us. *BioScience* 1973;23:343–352.

2. Wilson CB, Remington JS. What can be done to pre-

sent congenital toxoplasmosis? *Am J Obstet Gynecol* 1988; 158:337–363.

3. Henderson JB, Beattie CP, Hale EG, et al. The evaluation of new services: possibilities for preventing congenital toxoplasmosis. *Int J Epidemiol* 1984;13:63–72.

4. Alford CA. Immunoglobulin determinations in the diagnosis of fetal infection. *Pediatr Clin North Am* 1971;18:99–113.

5. Feldman HA. A nationwide serum survey of United states military recruits: 1962 VI. *Toxoplasma* antibodies. *Am J Epidemiol* 1963;81:385–391.

6. Kimball AC, Kean BH, Fuchs F. Congenital toxoplasmosis: a prospective study of 4,048 obstetric patients. *Am J Obstet Gynecol* 1971;111:211–218.

7. Remington JS, Desmonts G. Toxoplasmosis. In: Remington JS, Klein JO, eds. *Infectious diseases of the fetus and newborn infant*. 3rd ed. Philadelphia: WB Saunders Co, 1989;142–263.

8. Desmonts G, Couvreur J. Congenital toxoplasmosis: a prospective study of the offspring of 342 women who acquired toxoplasmosis during pregnancy. In: Thalhammer O, Baumgarten K, Pollak A, eds. *Perinatal medicine*. 6th European congress. Vienna. Stuttgart, George Thieme, 1978;51–60.

9. Jeannel D, Niel G, Costagliola D, et al. Epidemiology of toxoplasmosis among pregnant women in the Paris area. *Int J Epidemiol* 1988;17:895–902.

10. Decoster A, Darcy F, Caron A, et al. IgA antibodies against P30 as markers of congenital and acute toxoplasmosis. *Lancet* 1988;1:1064–1066.

11. Wilson M. Serologic aspects of toxoplasmosis. *J Am Vet Med Assoc* 1990;196:277–280.

12. Papoz L, Simondon F, Saimot W, et al. A simple model relevant to toxoplasmosis applied to epidemiologic results in France. *Am J Epidemiol* 1986;123:154–161.

13. Sever JL, Ellenberg JH, Ley AC, et al. Toxoplasmosis: maternal and pediatric findings in 23,000 pregnancies. *Pediatrics* 1988;82:181–192.

14. Frenkel JK, Ruiz A. Endemicity of toxoplasmosis in Costa Rica. *Am J Epidemiol* 1981;113:254–269.

15. Feldman HA, Miller LT. Serological study of toxoplasmosis prevalence. *Am J Hyg* 1956;64:320–325.

16. US Bureau of the Census. 1988 *statistical abstract of the United States*. 108th ed. Washington, DC, 1987.

17. Alford CA, Feft JW, Blankenship WJ, et al. Subclinical central nervous system disease of neonates: a prospective study of infants born with increased levels of IgM. *J Pediatr* 1969;75:1167–1178.

18. Couvreur J, Desmonts G, Tiercelin G, et al. Etude d'une série homogène de 210 cas de toxoplasmose congénitale chez des nourrissons âgés de 0 à 11 mois et dépistés de façon prospective. *Ann Pediatr* 1984;31:815–819.

19. Teutsch SM, Sulzer AJ, Ramsey JE, et al. *Toxoplasma gondii* isolated from amniotic fluid. *Obstet Gynecol* 1980;55 (Suppl 3):25–48.

20. Teutsch SM, Juranek DD, Sulzer A, et al. Epidemic toxoplasmosis associated with infected cats. *N Engl J Med* 1979;300:695–699.

21. Kimball AC, Kean BH, Fuchs F. The role of toxoplasmosis in abortion. *Am J Obstet Gynecol* 1971;111:219–226.

22. Eichenwald HF. A study of congenital toxoplasmosis. *Human Toxoplasmosis*. Copenhagen, Denmark: Williams & Wilkins Co, 1959;41–49.

23. Koppe JG, Kloosterman GJ. Congenital toxoplasmosis: a long term follow up. *Pediatr Padol* 1982;17:171–179.

24. Wilson CB, Remington JS, Stagno S, et al. Development of adverse sequelae in children born with subclinical congenital *Toxoplasma* infection. *Pediatrics* 1980;66:767–774.

25. Usher A, Chestnut LG, Violette DM. The value of reducing risks of death: a note on new evidence. *J Policy Anal Manage* 1989;8:88–102.

26. Landefeld JS, Seskin EP. The economic value of life: linking theory to practice. *Am J Public Health* 1982;72:555–566.

27. Bureau of Labor Statistics. Average weekly earnings. *Monthly Labor Review* 1989;112:85.

28. Bjerkedal T, Eriksson BI. Association of birth outcome with subsequent fertility. *Am J Obstet Gynecol* 1985; 147:569–624.

29. Peppers LG, Knapp RJ. Maternal reactions to fetal or infant death. *Psychiatry* 1980;43:155–159.

30. Tomasek RR. The grief of a mother physician on the death of her infant. *J Am Med Wom Assoc* 1988;43:31–37.

31. Conley RW. *The economics of mental retardation*. Baltimore and London: The John Hopkins University Press, 1973.

32. Rubin J, Berkowitz M, Moy R. *Economic consequences of severe disability*. Camden, NJ: Bureau of Economic Research, The State University of New Jersey, Rutgers, 1986;1–86.

33. Kakalik JS et al. *The cost of special education: summary of study findings*. Report R-2858-ED. Prepared under contract with the Office of Special Education and Rehabilitative Services. Santa Monica, Calif: The Rand Corp, 1981.

34. *Hospital statistics*. Chicago: American Hospital Association, 1987.

35. *New vaccine development: establishing priorities*. Washington, DC: Division of Health Promotion and Disease Prevention, Institute of Medicine, National Academy of Sciences, 1985.

36. Blacher J. Sequential stages of parental adjustment to the birth of a child with handicaps: fact or artifact? *Ment Retard* 1984;22:55–68.

37. Wikler LM. Periodic stresses of families of older mentally retarded children: an exploratory study. *Am J Ment Defic* 1986;90:703–706.

38. Fletcher RH, Fletcher SW, Wagner EH. *Clinical epidemiology—the essentials*. Baltimore: Williams & Wilkins Co, 1988;54–61.

39. Daffos F, Forestier F, Capella-Pavlovsky M, et al. Prenatal management of 746 pregnancies at risk for congenital toxoplasmosis. *N Engl J Med* 1988;318:271–275.

40. Frenkel JK. Congenital toxoplasmosis: prevention of pathogenesis. *Am J Obstet Gynecol* 1981;141:359–361.

41. Leighty JC. Strategies for control of toxoplasmosis. *J Am Vet Med Assoc* 1990;196:281–286.

42. A survey of the literature regarding the relationship between measures of IQ and income. Washington, DC: Environmental Protection Agency, 1984;1–31.

Trend in real starting salaries of US veterinary medical college graduates, 1980–1989

Mean starting salaries of US veterinary medical graduates have been reported annually since 1980.[1-5] The mean starting salary increased 29.1% from $17,175 in 1980 to $22,181 in 1988 (Table 1). This represents a compounded annual growth rate of 3.2%. The 1989 mean starting salary was $23,627, up 6.5% from 1988.

These data show that graduates' starting salaries have risen in money terms. In real terms, ie, correcting for the increased cost of living, graduates' mean starting salary declined. The most widely used measure of the change in the cost of living at the retail level is the Consumer Price Index for all urban consumers. The Consumer Price Index increased 49.9% from 1980 to 1989. This compares with the 37.6% increase in all graduates' mean starting salary from 1980 to 1989.

Analysis of graduates' starting salaries in real terms was accomplished by expressing the mean starting salary for each type of employer for each year, 1980 through 1989, in 1983 dollars. Table 1 shows mean starting salaries in money terms and real terms (1983 dollars).

Results show that in real terms, graduates' mean starting salary declined 10%, from $20,768 in 1980 to $18,699 in 1988, but increased by 1.9% from 1988 to 1989. Overall, the trend in real starting salaries has been flat since 1984. Current graduates have neither gained nor lost notable purchasing power relative to 1984 graduates.

Table 1—Real starting salaries of US veterinary medical graduates, 1980-1989

	Money income ($)				Real income (1983 dollars)			
Year	Private practice	Nonprivate practice	Advanced study	All graduates	Private practice	Nonprivate practice	Advanced study	All graduates
1980	18,552	17,967	12,033	17,175	22,433	21,725	14,550	20,768
1981	18,832	18,059	12,937	18,314	20,626	19,780	14,170	20,059
1982	19,351	20,864	13,781	18,700	19,970	21,531	14,222	19,298
1983	19,872	22,865	13,541	19,194	19,872	22,865	13,541	19,194
1984	20,237	21,493	13,797	19,527	19,403	20,607	13,228	18,722
1985	20,956	21,814	14,199	20,164	19,405	20,199	13,148	18,672
1986	21,205	22,027	15,186	20,478	19,275	20,022	13,804	18,615
1987	22,152	25,697	15,272	21,693	19,427	22,536	13,394	19,025
1988	23,079	25,832	15,766	22,181	19,455	21,776	13,291	18,699
1989	24,742	23,645*	16,363	23,627	19,959	19,074	13,200	19,060†

*Includes graduates who indicated university employment but who may, in fact, be pursuing advanced study. †Based on projected 4.4% increase in cost of living for 1989.

For the years 1986 to 1989, analysis focused on money incomes of male and female graduates entering full-time employment in private clinical practice. Mean starting incomes of male graduates have increased 17.6% from $21,576 in 1986 to $25,377 in 1989. Mean starting incomes of female graduates have increased 16.4% from $20,577 in 1986 to $23,944 in 1989. Figure 1 shows the trend in male and female graduates' mean starting salaries. In every year since 1986, male starting salaries have, on average, exceeded female starting salaries in private practice employment. Interestingly, since 1987, the amount by which the mean starting income of male graduates exceeded the mean starting income of female graduates increased from $236 in 1987, to

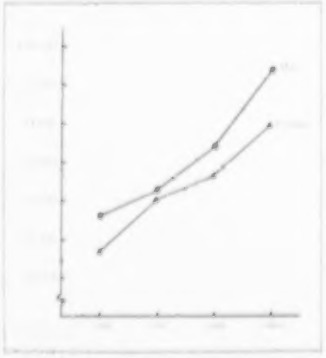


Figure 1—Trends in starting salaries of male and female veterinary medical college graduates entering private clinical practice.

Prepared by J. Karl Wise, PhD, Wisemark Associates, PO Box 681189, Schaumburg, IL 60168-1189.

$780 in 1988, to $1,433 in 1989.

References

1. Wise JK. Real starting salaries of US veterinary medical college graduates, 1978-1985. *J Am Vet Med Assoc* 1986; 188:427-428.

2. Wise JK. Employment, starting salaries, and educational indebtedness of 1986 graduates of US veterinary medical colleges. *J Am Vet Med Assoc* 1987; 190:209-210.

3. Wise JK. Employment, starting salaries, and educational indebtedness of 1987 graduates of US veterinary medical colleges. *J Am Vet Med Assoc* 1987; 191:1489-1490.

4. Wise JK. Employment, starting salaries, and educational indebtedness of 1988 graduates of US veterinary medical colleges. *J Am Vet Med Assoc* 1988; 193:1317-1318.

5. Wise JK. Employment, starting salaries, and educational indebtedness in 1989 graduates of US veterinary medical colleges. *J Am Vet Med Assoc* 1989; 195:1407-1408.

Book Review: Diagnostic Cytology of the Dog and Cat

The excellent, comprehensive presentation of cytologic sampling techniques, color photomicrographs, and substantial supporting text make *Diagnostic Cytology of the Dog and Cat* a definitive reference work for the student and experienced veterinary cytologist alike.

The first chapter is an in-depth review of cytologic techniques and terminology. The descriptions are clear and appropriately detailed. The authors discuss problems that arise with poor collection technique, poor slide making, and poor staining.

The focus of staining is a full-page table giving problems/solutions, with difficulties common to Romanowsky-type stains. Problems that occur with the commonly used supra-vital stain new methylene blue are not addressed. The discussion of stains might be more complete with mention of adjunct stains such as Gram, periodic-acid Schi*f*t reagent, and specific enzymatic stains.

The step-by-step illustrations of slide preparation are excellent. Algorithms are well laid out and informative and appear throughout the book. Tables contain good, concise information. The schematic drawings of cells and procedures are extremely helpful.

The greatest problem in the area of photomicrographs in the text is weak visual presentation and editing. Some of the photomicrographs have been poorly photographed or poorly reproduced, and cellular detail is lacking. The stain used and magnification of the photographs are inconsistently listed. The text and photomicrographs sometimes are "out of sync," with photomicrographs preceding text description by as much as 4 pages. When the reader is referred to a plate, the specific figure is not given. The use of arrows and other labels to specifically identify described morphology is grossly underused, which is disappointing, because the overall quality of the book suggests that the authors intended its usefulness for students and experienced practitioners.

Because the authors favor a systems approach, the table of contents reveals an organizational weakness. Topics one might expect to be discussed in close proximity are separated by discussions of other organ systems. For example, Chapter 8 covers the lymph nodes and Chapter 17 discusses the splenic parenchyma. Cross-referencing to other chapters avoids repetition, but not always successfully.

The index is inconsistent. For example, although mycotic agents are listed only under the general heading of "Infectious agents," parasites are listed individually, under "Infectious agents" where appropriate and under the heading of "Parasitism" also. Terms somewhat specific to cytology, like lymphoglandular bodies, are not indexed, although they are discussed and photographed on page 95.

This high-caliber cytologic text is reasonably priced and belongs in any veterinary library, especially a working library. Although there are some organizational flaws, they are quickly overridden by the comprehensive coverage of cytologic samples encountered in veterinary medicine.—*[Diagnostic Cytology of the Dog and Cat. Edited by Rick L. Cowell and Ronald D. Tyler. 259 pages; illustrated. American Veterinary Publications Inc, 5782 Thornwood Dr, Goleta, CA 93117. 1989. Price $75.45.]*—LAURIE G. O'ROURKE